of prevalent and recurrent cases as well incident cases. However, our concern with this possible bias at the outset of the study led us to exclude all patients with a history of previous genital warts. This included those previously diagnosed at SSHC, and those who gave a history of having their warts managed elsewhere. Consequently, when we state a new diagnosis of genital warts, this is precisely what we mean.

With regard to the conduct of the study, this was performed with the assistance of the current data manager responsible for the SSHC data base, whose help and assistance were duly acknowledged.

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Photosensitivity reaction to efavirenz

EDITOR,—The non-nucleoside reverse transcriptase inhibitor (NNRTI) efavirenz is a recent addition to the armamentarium available to physicians in the treatment of HIV infection. However, at present the known side effect profile of this new agent is still in its infancy. We would like to report a case of photosensitivity associated with efavirenz.

A 27 year old white homosexual man was commenced on combivir (zidovudine/lamivudine) and efavirenz in March of 1999. One month later he reported that he was well and had no major side effects associated with his new combination. However, 4 weeks further into treatment he represented with an itchy rash affecting his arms and hands. On examination there was a maculopapular rash over the affected area but there was no oral ulceration, conjunctivitis, or fever. A drug reaction was diagnosed and he was prescribed antihistamines and asked to continue with his medication. One week later the rash had subsided. Then having spent a day outside in the sun he had a florid recurrence of the rash over the exposed areas (arms, back of neck, face, and ears). The rash was significantly worse over his elbows where there was obvious blistering and oedema. His medication was stopped and 3 weeks later the rash had completely resolved. Hepatitis C antibody and porphyria screening were negative. This man had been diagnosed as HIV antibody positive in June 1997. In March 1998 his viral load was 356 790 copies/ml (Roche PCR) and his CD 4 count was 512×10^6 cells/l, he was commenced on dual antiretroviral therapy with stavudine and didanosine (patient choice). Initially he did very well as the viral load became undetectable (<400 copies/ml). However, after 9 months on this combination his viral load began to rebound (5192 copies/ml) and a change in antiretroviral therapy was initiated to combivir and nevirapine which he initiated in the normal way (dose escalation at 2 weeks of nevirapine). He was started on this combination as he wished to take a protease sparing regimen. However, 1 week later he developed a rash affecting his entire body, especially his trunk and arms, associated with enlarged lymph nodes and constitutional symptoms, fever, and lethargy. In view of the constitutional symptoms it was decided to stop this present combination. One month later, the rash had settled, he then commenced combivir and efavirenz.

Photosensitivity in the context of HIV has been reported as a presenting sign of underlying HIV infection in a number of cases.[1–3] In addition to this porphyria cutanea tarda (PCT) has been reported in the context of HIV infection and has been associated with concomitant hepatitis C infection[4]; however, screening for both these conditions was negative. Switching from nevirapine to efavirenz in this context may have been regarded as unwise; however, of 19 patients who have been intolerant of nevirapine secondary to the development of rash, who have switched to efavirenz only nine have developed a mild to moderate rash, of which only two needed to discontinue therapy.[5] Photosensitivity in the context of HIV infection may not only be a presenting condition but also secondary to concomitant treatment.

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HIV associated cytomegalovirus retinitis in Melbourne, Australia

EDITOR,—We report the results of a 12 year review of human immunodeficiency virus (HIV) associated cytomegalovirus (CMV) retinitis in Melbourne, Australia.

We conducted a retrospective review of all HIV infected patients diagnosed with CMV retinitis at Fairfield Hospital and the Alfred Hospital between 1984 and 1996, aiming to identify factors at diagnosis of CMV retinitis which were predictive of outcome. Both hospitals had the same protocol for the treatment of CMV retinitis and employed 3 monthly ophthalmological screening of all HIV infected patients with CD4 counts of less than 50×10^6/l.

The study outcomes were visual loss and death. Moderate visual loss was defined as a visual acuity of less than 6/12 in the better eye, and severe visual loss as visual acuity of less than 6/60 in the better eye (this is legal blindness in Australia).

CMV retinitis was diagnosed in 212 of 1281 patients (16.5%) with AIDS over the study period. As of June 1998, 193 (93%) had died, at a median time of 36 weeks (range 0–192) from CMV diagnosis. Seventy four patients (35%) developed moderate visual loss at a median time of 23 weeks (range 0–163) and 30 patients (14%) developed severe visual loss at a median time of 35 weeks (range 0–120) from diagnosis of CMV retinitis.

The presence of visual symptoms at diagnosis of CMV retinitis was predictive of the development of moderate visual loss (relative risk 2.1, 95% confidence interval 1.1–4.2). Fifty eight of 138 patients (42%) with visual symptoms at diagnosis developed moderate visual loss, compared with 16 of 64 patients (25%) who were asymptomatic at diagnosis (p=0.02). The presence of visual symptoms at diagnosis was not predictive of the development of severe visual loss, or early death (p>0.2). Other factors measured at diagnosis of CMV retinitis included the patients' age, CD4 count, weight, visual acuity, and the presence of any previous AIDS defining condition. None of these was associated with the development of visual loss or early death (p>0.1).

The advent of highly active antiretroviral therapy (HAART) has resulted in a reduction in the incidence of new diagnoses of opportunistic infections. Prolonged survival times with CMV retinitis have been demonstrated in patients who achieve immunological recovery with HAART.[1 2] The ability to predict those patients who are at highest risk of visual loss may assist in advising those who may reasonably cease maintenance therapy for CMV retinitis following immune restoration. An understanding of the natural history of CMV retinitis in the pre-HAART years remains important in managing patients who are failing HIV therapy.

The only factor measurable at diagnosis of CMV retinitis that was predictive of outcome was the presence of visual symptoms. The use of routine ophthalmological screening in HIV infected individuals with low CD4 counts aims to detect CMV retinitis before visual symptoms occur. It is possible that visual loss may be prevented by detecting disease before retinal damage occurs. A prospective evaluation is needed to confirm this finding.

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